i. The trifloxystrobin toxicology database is complete for FQPA assessment.

ii. There is no indication of increased susceptibility of rat or rabbits to trifloxystrobin.

In the developmental and reproductive toxicity studies, effects in the fetuses/offspring were observed only at or above treatment levels which resulted in evidence of parental toxicity.

Using the same exposure assumptions as employed for the determination in the general population, it has been calculated that the percent of the RfD that will be utilized by aggregate exposure to residues of trifloxystrobin is <2.0% for non-nursing infants (<1 year old)(the most impacted sub-population). Therefore, based on the completeness and reliability of the toxicity data base and the conservative exposure assessment, Bayer concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to trifloxystrobin residues.

F. International Tolerances

No Codex MRLs have been established for residues of trifloxystrobin.

[FR Doc. 01–22025 Filed 8–30–01; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[PF–1041; FRL–6796–1]

Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of a pesticide petition proposing the establishment of regulations for residues of a certain pesticide chemical in or on various food commodities.

DATES: Comments, identified by docket control number PF–1041, must be received on or before October 1, 2001.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the **SUPPLEMENTARY INFORMATION.** To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–1041 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: James A. Tompkins, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 305–5697; e-mail address: tompkins.jim@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be affected by this action if you are an agricultural producer, food manufacturer or pesticide manufacturer. Potentially affected categories and entities may include, but are not limited to:

Categories	NAICS codes	Examples of potentially affected entities
Industry	111 112 311 32532	Crop production Animal production Food manufacturing Pesticide manufacturing

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in the table could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether or not this action might apply to certain entities. If you have questions regarding the applicability of this action to a particular entity, consult the person listed under **FOR FURTHER INFORMATION CONTACT.**

B. How Can I Get Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically*. You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations,'' ''Regulations and Proposed Rules,'' and then look up the entry for this document under the ''**Federal Register**—Environmental Documents.'' You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person*. The Agency has established an official record for this action under docket control number PF–1041. The official record consists of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as confidential business information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

C. How and to Whom Do I Submit Comments?

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–1041 in the subject line on the first page of your response.

1. *By mail*. Submit your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460.

2. *In person or by courier*. Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA. The PIRIB is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

3. *Electronically*. You may submit your comments electronically by e-mail to: opp-docket@epa.gov, or you can submit a computer disk as described above. Do not submit any information electronically that you consider to be CBI. Avoid the use of special characters and any form of encryption. Electronic submissions will be accepted in Wordperfect 6.1/8.0 or ASCII file format. All comments in electronic form must be identified by docket control number PF–1013. Electronic comments

may also be filed online at many Federal Depository Libraries.

D. How Should I Handle CBI That I Want to Submit to the Agency?

Do not submit any information electronically that you consider to be CBI. You may claim information that you submit to EPA in response to this document as CBI by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public version of the official record. Information not marked confidential will be included in the public version of the official record without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person identified under **FOR FURTHER INFORMATION CONTACT**.

E. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

3. Provide copies of any technical information and/or data you used that support your views.

4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.

5. Provide specific examples to illustrate your concerns.

6. Make sure to submit your comments by the deadline in this notice.

7. To ensure proper receipt by EPA, be sure to identify the docket control number assigned to this action in the subject line on the first page of your response. You may also provide the name, date, and **Federal Register** citation.

II. What Action is the Agency Taking?

EPA has received a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of a certain pesticide chemical in or on various food commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that this petition contains data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

List of Subjects

Environmental protection, Agricultural commodities, Feed additives, Food additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: August 21, 2001.

Donald R. Stubbs, Acting

Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner summary of the pesticide petition is printed below as required by section 408(d)(3) of the FFDCA. The summary of the petition was prepared by the petitioner and represents the view of the petitioners. EPA is publishing the petition summary verbatim without editing it in any way. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

Gowan Company and Interregional Research Project # 4

PP 0F6169, 1F6229 and 0E6206

This notice announces the initial filing of pesticide petitions proposing the establishment of regulations for residues of a certain pesticide chemical in or on various food commodities. EPA has received pesticide petitions (PP 0F6169 and 1F6229) from Gowan Company, Yuma, AZ, 85364, and (PP 0E6206) from the Interregional Research Project #4, 681 U.S. Highway No.1 South, North New Brunswick, NJ 08902–3390, proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing tolerances for residues of the herbicide halosulfuron-methyl (methyl 5-[(4,6-dimethoxy-2-pyrimidinyl)amino] carbonyl aminosulfonyl-3-chloro-1-methyl-1H-pyrazole-4-carboxylate) in or on the fruiting vegetables (excluding cucurbits) Crop Group 8 at 0.05 parts per million (ppm) (PP 0F6169), asparagus at 0.8 ppm (PP 1F6229), and the melon subgroup Subgroup 9A at 0.1 ppm. EPA has determined that the petitions contain data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petitions. Additional data may be needed before EPA rules on the petitions.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of halosulfuron-methyl as well as the nature of the residues in plants is adequately understood for purposes of these tolerances. Metabolism studies were conducted in three crops, viz.; field corn, sugarcane and soybeans. Metabolism depends on the mode of application. Preemergent applications result in rapid soil degradation of halosulfuron-methyl followed by crop uptake of the resulting pyrazole moiety. The pyrimidine ring binds tightly to soil and is eventually converted to carbon dioxide by microbial degradation. In postemergent applications, little metabolism and translocation take place resulting in unmetabolized parent compound as the major residue on the directly treated foliar surfaces. Very low residue levels of the metabolite 3-chloro-1-methyl-5-sulfamoylpyrazole-4-carboxylic acid (3-CSA) are found in the grain.

2. *Analytical method*. A practical analytical method, gas chromatography with a nitrogen specific detector (TSD) which detects and measures residues of halosulfuron-methyl, is available for enforcement purposes with a limit of detection that allows monitoring of food with residues at or above the levels set in these tolerances. This enforcement method has been submitted to the Food and Drug Administration for publication in the Pesticide Analytical Manual (PAM II). It has undergone independent laboratory validation and validation at the Beltsville laboratory. An Analytical Chemistry section of the EPA concluded that the method is adequate for enforcement. The analytical method is also available for analyzing meat by-products, which also underwent successful independent laboratory and Beltsville laboratory validations.

3. *Magnitude of residues*. In asparagus residue studies, the magnitude of the residues found in the raw agricultural commodity (RAC) was less than 0.8 ppm using an analytical method with limit of quantitation (LOQ) of 0.05 ppm; residues in cantaloupe were less than 0.1 ppm. In tomato and pepper residue studies, there were no quantifiable residues found in the RACs. There were also no detectable residues at a LOQ of 0.05 ppm found in tomato processed commodities at treatment rates of more than 2 times the maximum recommended rate per season.

B. Toxicological Profile

1. *Acute toxicity*. Acute toxicological studies placed the technical-grade halosulfuron-methyl in Toxicity Category III. A 90–day feeding study in rats resulted in a lowest observed adverse effect level (LOAEL) of 497 milligrams/kilograms/day (mg/kg/day) in males and 640 mg/kg/day in females, and a no observed adverse effect level (NOAEL) of 116 mg/kg/day in males and 147 mg/kg/day in females.

2. *Genotoxicity*. Bacterial/mammalian microsomal mutagenicity assays were performed and found not to be mutagenic. Two mutagenicity studies were performed to test gene mutation and found to produce no chromosomal aberrations or gene mutations in cultured Chinese hamster ovary cells. An *in vivo* mouse micronucleus assay did not cause a significant increase in the frequency of micronucleated polychromatic erythrocytes in bone marrow cells. A mutagenicity study was performed on rats and found not to induce unscheduled DNA synthesis in primary rat hepatocytes.

3. *Reproductive and developmental toxicity*. A developmental toxicity study in rats resulted in a developmental LOAEL of 750 mg/kg/day, based on decreases in mean litter size and fetal body weight, and increases in resorptions, resorptions/dam, post-implantation loss and in fetal and litter incidences of soft tissue and skeletal variations, and a developmental NOAEL of 250 mg/kg/day. Maternal LOAEL was 750 mg/kg/day based on increased incidence of clinical observations, reduced body weight gains, and reduced food consumption and food efficiency. The maternal NOAEL was 250 mg/kg/day.

A developmental toxicity study in rabbits resulted in a developmental LOAEL of 150 mg/kg/day, based on decreased mean litter size and increases in resorptions, resorptions/dam and post-implantation loss, and a developmental NOAEL of 50 mg/kg/day. The maternal LOAEL was 150 mg/kg/day based on reduced body weight gain and reduced food consumption and food efficiency. The maternal NOAEL was 50 mg/kg/day.

A dietary 2–generation reproduction study in rats resulted in parental toxicity at 223.2 mg/kg/day in males and 261.4 mg/kg/day in females in the form of decreased body weights, decreased body weight gains, and reduced food consumption during the premating period. Very light effects were noted in body weight of the offspring at this dose. This effect was considered to be developmental toxicity (developmental delay) rather than a reproductive effect. No effects were noted on reproductive or other developmental toxicity parameters. The systemic/ developmental toxicity LOAEL was 223.2 mg/kg/day in males and 261.4 mg/kg/day in females; the systemic/developmental toxicity NOAEL was 50.4 mg/kg/day in males and 58.7 mg/kg/day in females. The reproductive LOAEL was greater than 223.2 mg/kg/day in males and 261.4 mg/kg/day in females; the reproductive NOAEL was equal to or greater than 223.2 mg/kg/day in males and 261.4 mg/kg/day in females.

4. *Subchronic toxicity.* A 21–day dermal toxicity study in rats resulted in a NOAEL of 100 mg/kg/day in males and greater than 1,000 mg/kg/day in females. The only treatment-related effect was a decrease in body weight gain of the 1,000 mg/kg/day group in males.

5. *Chronic toxicity*. A 1–year chronic oral study in dogs resulted in a LOAEL of 40 mg/kg/day based on decreased weight gain and a NOAEL of 10 mg/kg/day for systemic toxicity. A 78–week carcinogenicity study was performed on mice. Males in the 971.6 mg/kg/day group had decreased body weight gains and an increased incidence of microconcretion/mineralization in the testis and epididymis. No treatment-related effects were noted in females. Based on these results, a LOAEL of 971.9 mg/kg/day was established in males and NOAELs of 410 mg/kg/day in males and 1,214.6 mg/kg/day in females were established. The study showed no evidence of carcinogenicity. A combined chronic toxicity/ carcinogenicity study in rats resulted in a LOAEL of 225.2 mg/kg/day in males and 138.6 mg/kg/day in females based on decreased body weight gains, and a NOAEL of 108.3 mg/kg/day in males and 56.3 mg/kg/day in females. The study showed no evidence of carcinogenicity.

6. *Animal metabolism*. EPA stated that the nature of the residue in ruminants was determined to be adequately understood. In the tissues and milk of goats, the major extractable residue was the unmetabolized parent compound. Based on the low residues of the parent compound in corn grain and the low transfer of residues in the metabolism study, tolerances on poultry products were not required. In the rat metabolism study, parent compound was absorbed rapidly but incompletely. Excretion was relatively rapid at all doses tested with majority of radioactivity eliminated in the urine and feces by 72 hours. Fecal elimination of parent was apparently the result of unabsorbed parent.

7. *Metabolite toxicology*. The toxicology studies listed below were conducted with the 3–CSA metabolite. Based on the toxicological data of the 3–CSA metabolite, EPA concluded that it has lower toxicity compared to the parent compound and that it should not be included in the tolerance expression. The residue of concern is the parent compound only.

i. A 90–day rat feeding study resulted in a LOAEL in males of >20,000 ppm and a NOAEL of 20,000 ppm (1,400 mg/kg/day). In females, the LOAEL is 10,000 ppm (772.8 mg/kg/day) based on decreased body weight gains and a NOAEL of 1,000 ppm (75.8 mg/kg/day).

ii. A developmental toxicity resulted in a LOAEL for maternal toxicity of 1,000 mg/kg/day based on the absence of systemic toxicity, a NOAEL of 1,000 mg/kg/day. The developmental LOAEL is >1,000 mg/kg/day and the NOAEL is 1,000 mg/kg/day

iii. The microbial reverse gene mutation did not produce any mutagenic effect while the mammalian cell gene mutation/Chinese hamster ovary cells did not show a clear evidence of mutagenic effect in the Chinese hamster ovary cells.

iv. The mouse micronucleus assay did not show any clastogenic or aneugenic effect.

8. *Endocrine disruption.* No specific tests have been conducted with halosulfuron-methyl to determine whether the chemical may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen or other endocrine effects. However, there were no significant findings in other relevant toxicity tests, i.e., teratology and multi-generation reproduction studies, which would suggest that halosulfuron-methyl produces effects characteristic of the disruption of the estrogenic hormone.

C. Aggregate Exposure

1. *Dietary exposure.* Tolerances have been established (40 CFR 180.479) for residues of halosulfuron-methyl in or on a variety of plant and animal RACs including field corn, grain sorghum (milo), sweet corn (kernel + cobs with husks removed), pop corn grain, sugarcane cane, tree nuts nutmeat, pistachio nuts nutmeat, cotton undelinted seed, and rice grain at 0.05 ppm; squash/cucumber subcrop group 9B at 0.5 ppm; and secondary tolerances in meat and meat by-products at 0.1 ppm (cattle, goats, hogs, horses, and sheep). Additional tolerances are being requested by Gowan for fruiting vegetables (except cucurbits) crop group

8 at 0.05 ppm and asparagus at 0.8 ppm, and by IR-4 for the melon subcrop group 9A at 0.1 ppm.

Food—a. Acute exposure. The acute Reference Dose (aRfD) for halosulfuron-methyl is 0.5 mg/kg/day. For purposes of assessing the potential dietary exposure from food under existing and proposed tolerances, aggregate exposure is based on the Theoretical Maximum Residue Contribution (TMRC) which is an estimate of the level of residues consumed daily if each food item contained pesticide residues equal to the tolerance. The calculated TMRC value using the 99.9th percentile consumption data was 0.006 mg/kg body weight/day for the general U.S. population. This value utilizes only 1.2% of the aRfD for all established and proposed tolerances for halosulfuron-methyl. TMRC is obtained by multiplying the tolerance levels for each commodity by the daily consumption of the food forms of that commodity eaten by the U.S. population and various population subgroups. In conducting this exposure assessment, conservative assumptions were made, e.g., 100% of all commodities will contain halosulfuron-methyl residues and those residues would be at the level of their respective tolerances. This results in a large overestimate of human exposure. Food consumption data from DEEM software (Novigen Sciences, Inc.) were used in the calculation. Field corn and sorghum forage and fodder are fed to animals, thus exposure of humans to residues from these commodities might result if such residues are transferred to meat, milk, poultry or eggs. However, based on the results of animal metabolism and the amount of halosulfuron-methyl expected in animal feeds, it can be concluded that there is no reasonable expectation that residues of halosulfuron-methyl will exceed existing tolerances in meat.

b. *Chronic exposure.* The chronic Reference Dose (cRfD) is 0.1 mg/kg/day. The calculated TMRC value using 99.9th percentile consumption data was 0.000779 mg/kg body weight/day for children 1–6 years, the most exposed subpopulation group. This value utilizes only 0.8% of the CRfD for all established and proposed tolerances for halosulfuron-methyl.

c. *Short-term and intermediate-term exposure.* The short-term NOAEL for females 13 + years and infants and children is 50 mg/kg/day. Comparing the NOAEL with the chronic food exposure from DEEM analysis of 0.00042 mg/kg/day for females 13+ and 0.00090 mg/kg/day for infants and children results in food MOEs of 119,000 and 55,600, respectively. The intermediate-term NOAEL is 10 mg/kg/day, comparing the NOAEL with the chronic food exposure from DEEM analysis of 0.00090 mg/kg/day for children (1–6 years old) results in a food MOE of 11,100.

d. *Chronic risk-carcinogenic.* Halosulfuron-methyl has been classified as a Group E chemical based upon the lack of evidence of carcinogenicity in mice and rats, and has been classified as a not likely human carcinogen.

e. *Drinking water.* There is no Maximum Contaminant Level (MCL) established for residues of halosulfuron-methyl. It is not listed for MCL development or drinking water monitoring under the Safe Drinking Water Act nor is it a target of EPA's National Survey of Wells for Pesticides. Gowan and IR-4 are not aware of any halosulfuron-methyl detections in any wells, ponds, lakes or streams resulting from its use in the United States. The estimated drinking water environmental concentrations (DWEC) in ground water (acute and chronic) is 0.008 µg/L. The estimated DWECs (acute and chronic) for surface water are 4.3 µg/L and 1.1 µg/L, respectively. These estimates are based on a maximum application rate of 0.063 lbs active per acre which may be applied twice per season.

f. *Acute exposure and risk.* Acute drinking water levels of concern (DWLOCs) have been calculated for exposure to halosulfuron-methyl in drinking water for the relevant population subgroups of females 13+ years and infants and children. The acute DWLOC is 15,000 µg/L for females 13+ years and 5,000 µg/L for infants and children. The calculated DWLOCs are significantly higher than the DWECs for ground water (0.008 µg/L) and surface water (4.3 µg/L).

g. *Chronic exposure and risk.* Chronic DWLOCs have been calculated for exposure to halosulfuron-methyl in drinking water for the U.S. population (48 states) and the relevant subgroups of females 13+ years and infants and children. The chronic DWLOC is 3,500 µg/L for the U.S. population, 3,000 µg/L for females 13+ years, and 1,000 µg/L for infants and children. The calculated DWLOCs are significantly higher than the DWECs for ground water (0.008 µg/L) and surface water (1.1 µg/L).

h. *Short and intermediate term exposure and risk.* Short-term and intermediate-term DWLOCs have been calculated for exposure to halosulfuron-methyl in drinking water for the relevant population subgroups. The short-term DWLOC is 10,000 µg/L for females 13+ years and 3,700 µg/L for infants and children. The intermediate-term DWLOC is 590 µg/L for adult males, 57 µg/L for females 13+ years, and 160 µg/L for infants and children. The calculated intermediate-term DWLOCs are significantly higher than the chronic DWECs for surface water (1.1 µg/L). The calculated short-term DWLOCs are significantly higher than the acute DWECs for ground water (0.008 µg/L) and surface water (4.3 µg/L).

i. *Conclusion.* EPA has concluded that potential levels of halosulfuron-methyl in soil and water do not appear to have significant toxicological effects on humans or animals and presents a negligible risk. Based on the very low level of mammalian toxicity, lack of other toxicological concerns and low use rates, there is reasonable certainty that no harm will result from exposure to halosulfuron-methyl via drinking water sources.

2. *Non-dietary exposure.* Halosulfuron-methyl is labeled for use on commercial and residential turf and other non-crop sites. For residential applicators, short-term and intermediate-term exposure may occur. Chronic exposure (>6 months of continuous exposure) are not expected.

i. *Acute exposure and risk.* There is potential for exposure to halosulfuron-methyl by homeowner. However, since endpoints for acute dermal or inhalation were not identified, the use of halosulfuron-methyl on residential non-food sites is not expected to pose an unacceptable acute risk.

ii. *Chronic exposure and risk.* Chronic exposures for residential use of halosulfuron-methyl are not expected and a chronic non-deitary endpoint was not identified, therefore, the use on residential non-food sites is not expected to pose an unacceptable chronic risk.

iii. *Short-term and intermediate-term exposure and risk.*re is potential for short-term or intermediate-term dermal exposure to residential handlers, therefore residential exposure assessments were conducted to assess the following post-application exposure scenarios: (a) Dermal exposure to residues on turf; (b) children's incidental non-dietary ingestion of residues on residential lawn from hand-to-mouth transfer; and (c) children's ingestion of pesticide-treated turfgrass.

The short-term dermal MOE for residential handlers is 4,200 which is significantly greater than the minimum acceptable MOE of 100.

The short-term dermal MOE for exposure from treated lawns for adult males, adult females, and children are 390, 330, and 420, respectively, which are significantly greater than the

minimum acceptable MOE of 100. The intermediate-term dermal MOE for exposure from treated lawns for adult males, adult females, and children are 120, 100, and 130, respectively, which are significantly greater than the minimum acceptable MOE of 100. Therefore the use of halosulfuron-methyl on residential non-food sites is not expected to pose an unacceptable short-term or intermediate-term risk.

The short-term and intermediate- term oral MOE for hand-to-mouth transfer for children are 4,900 and 1,500, respectively, which are significantly greater than the minimum acceptable MOE of 100. Therefore, the use of halosulfuron-methyl on residential non-food sites is not expected to pose an unacceptable short-term or intermediate-term risk.

The short-term and intermediate-term oral MOE for incidental ingestion by children are 210,000 and 66,000, respectively, which are significantly greater than the minimum acceptable MOE of 100. Therefore, the use on residential non-food sites is not expected to pose an unacceptable short-term or intermediate- term risk.

D. Cumulative Effects

Halosulfuron-methyl belongs to the sulfonyl urea class of chemistry. The mode of action of halosulfuron-methyl is the inhibition of the plant enzyme aceto lactase synthetase (ALS), which is essential for the production of required amino acid in plants. Although other registered sulfonyl ureas may have similar herbicidal mode of action, there is no information available to suggest that these compounds exhibit a similar toxicity profile in the mammalian system that would be cumulative with halosulfuron-methyl. Thus, consideration of a common mechanism of toxicity is not appropriate at this time. Gowan is considering only the potential risks of halosulfuron-methyl in its aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*—i. *Acute risk.* Aggregate exposure risk includes exposure from food and water. The risk from acute ''food only'' exposure is less than 2.9% of the RfD for all population groups which is less than the EPA's level of concern. The lowest DWLOC calculated was 5,000 µg/L for infants and children. The calculated DWLOC for females (13+ years) was 15,000 µg/L. For both subgroups, the DWLOC is significantly higher than the DWEC for acute ground water (0.008 g/L) and surface water (4.3 µg/L), therefore, the risk from aggregate exposure to halosulfuron-methyl residues from all anticipated dietary exposure routes does not pose appreciable risks to human health.

ii. *Chronic risk.* Aggregate chronic exposure to halosulfuron-methyl from ''food only'' utilizes less than 1% of the RfD for the most sensitive subgroup, children (1–6 years). The lowest DWLOC calculated was 1,000 µg/L for infants and children which is significantly higher than the DWEC for chronic ground water (0.008 g/L) and surface water (1.1 µg/L). Therefore, the aggregate risk from chronic exposure to halosulfuron-methyl residues from all anticipated dietary exposures does not pose appreciable risks to human health.

iii. *Short-term and intermediate-term risk.*

a. Short-term aggregate exposure takes into account chronic dietary food and water plus short-term residential exposure. For halosulfuron-methyl, the EPA has determined that it is appropriate to aggregate exposure via oral exposure route (food and water) with those via oral and dermal exposure routes from residential uses. The MOEs for ''food only'' and residential exposure routes are 13,859 and 310 for females 13+ years. Short-term DWLOC for females 13+ is 10,000 mu;g/L which is substantially higher than the DWEC for acute surface water (4.3 µg/L). The food only and residential (oral and dermal) MOEs are well above the acceptable short-term aggregate MOE of 100. Therefore, exposure to halosulfuron-methyl residues resulting from current and proposed uses does not pose a short-term aggregate risk.

b. Intermediate-term aggregate exposure takes into account chronic dietary food and water plus intermediate-term residential exposure. The MOEs for ''food only'' and residential exposure routes are 24,000 and 120 for adult males, and 23,800 and 100 for females 13+ years. The intermediate-term DWLOCs are 590 µg/L and 57 µg/L, respectively, for adult males and females 13+. Intermediate-term DWLOCs are substancially higher than the DWEC for chronic surface water (1.1 µg/L). The food only and residential (dermal) MOEs are above the acceptable short-term aggregate MOE of 100. Therefore, exposure to halosulfuron-methyl residues resulting from current and proposed uses does not pose a intermediate-term aggregate risk.

c. Aggregate cancer risk. Halosulfuron-methyl has been classified as a Group E chemical based upon the lack of evidence of carcinogenicity in mice and rats, and has been classified as a not likely human carcinogen.

d. Conclusion. Based upon these risk assessments, Gowan concluded that there is a reasonable certainty that no harm will result from aggregate exposure to halosulfuron-methyl residues resulting from current and proposed uses.

2. *Infants and children.*—i. *Safety factor.* FFDCA section 408 provides that EPA may apply an additional safety factor (up to 10) in the case of threshold effects for infants and children to account for pre-natal and post-natal toxicity and the completeness of the data base. Except for the pending request for a developmental neurotoxicity study, the toxicity data base is complete for halosulfuron-methyl. Based upon reliable toxicity data, the use of an additional 10x safety factor is not warranted. Dietary assessments do not indicate a level of concern for potential risks to infants and children based upon the low use rates of halosulfuron-methyl and that the results of field and animal RAC studies conclude that detectable residues are not expected in human foods.

ii. *Acute risk.* The acute RfD was determined to be 0.5 mg/kg/day based upon the developmental rabbit study. The percent of the RfD occupied is 2.9% for the most sensitive population subgroup, nursing infants (<1 year). The drinking water level of comparison (DWLOC) for acute exposure for infants and children is 5,000 µg/L and is significantly greater than the maximum concentration of halosulfuron-methyl in drinking water (0.008 µg/L in ground water and 4.3 µg/L in surface water).

iii. *Chronic risk.* The chronic RfD was determined to be 0.1 mg/kg/day based upon the chronic dog study. The percent of RfD occupied is 0.9% for the most sensitive subgroup, children (1–6 years old). The DWLOC for chronic exposure for infants and children is 1,000 µg/L and is significantly greater than the maximum concentration of halosulfuron-methyl in drinking water (0.008 µg/L in ground water and 1.1 µg/L in surface water).

iv. *Short-term and intermediate-term risk.* An aggregate exposure estimate and risk assessment was calculated for post-application exposure to halosulfuron-methyl from treated lawns. Short-term MOEs for food, residential oral, and residential dermal are 55,600, 4,900, and 420, respectively, for infants and children. Intermediate-term MOEs for food, residential oral, and residential dermal are 11,100, 1,500, and 130, respectively, for children and infants. The short-term and intermediate-term DWLOCs for infants and children were 3,700 and 160 mu;g/L, respectively, which are substancially higher than the

DWECs for acute surface water (4.3 µg/L) and chronic surface water (1.1 µg/L).

v. *Conclusion.* Therefore, based on complete and reliable toxicity data and the conservative exposure assessment, Gowan concludes that there is reasonable certainty that no harm will result to infants and children from aggregate exposure to halosulfuron-methyl residues with respect to the proposed new uses.

F. International Tolerances

Maximum residue levels have not been established for residues of halosulfuron-methyl on any food or feed crop by the Codex Alimentarius Commission.

[FR Doc. 01–22024 Filed 8–30–01; 8:45 am]

BILLING CODE 6560–50–S

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Notice of Meeting of the Advisory Committee on Minority Health

AGENCY: Office of the Secretary, Office of Public Health and Science, Office of Minority Health.

ACTION: Notice is given of the third meeting.

The Advisory Committee on Minority Health will meet on Thursday, September 20, 2001 from 9 a.m. to 5 p.m., and Friday, September 21, 2001, from 8:30 a.m.–3 p.m. The meeting will be held at the Holiday Inn Georgetown, Mirage I Room, 2101 Wisconsin Avenue, NW., Washington, DC.

The Advisory Committee will discuss racial and ethnic disparities in health, as well as, other related issues.

The meeting is open to the public. There will be an opportunity for public comment which will be limited to five minutes per speaker. Individuals who would like to submit written statements should mail or fax their comments to the Office of Minority Health at least two business days prior to the meeting.

For further information, please contact Ms. Patricia Norris, Office of Minority Health, Rockwall II Building, 5515 Security Lane, Suite 1000, Rockville, Maryland 20852. Phone: 301–443–5084 Fax: 301–594–0767.

Dated: August 23, 2001.

Nathan Stinson, Jr.,

Deputy Assistant Secretary for Minority Health.

[FR Doc. 01–21976 Filed 8–30–01; 8:45 am]

BILLING CODE 4150–29–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Notice of Publication of the Executive Summary of the report, ''Ethical and Policy Issues in Research Involving Research Participants'', by the National Bioethics Advisory Commission (NBAC)

SUPPLEMENTARY INFORMATION: The President established the National Bioethics Advisory Commission (NBAC) on October 3, 1995 by Executive Order 12975 as amended. The functions of NBAC are as follows:

(a) Provide advice and make recommendations to the National Science and Technology Council and to other appropriate government entities regarding the following matters:

(1) The appropriateness of departmental, agency or other governmental programs, policies, assignments, missions, guidelines, and regulations as they relate to bioethical issues arising from research on human biology and behavior; and

(2) applications, including the clinical applications, of that research.

(b) Identify broad principles to govern the ethical conduct of research, citing specific projects only as illustrations for such principles.

(c) Shall not be responsible for the review and approval of specific projects.

(d) In addition to responding to requests for advice and recommendations from the National Science and Technology Council, NBAC also may accept suggestions of issues for consideration from both the Congress and the public. NBAC may also identify other bioethical issues for the purpose of providing advice and recommendations, subject to the approval of the National Science and Technology Council. The members of NBAC are as follows:

Harold T. Shapiro, Ph.D., Chair
Patricia Backlar
Arturo Brito, M.D.
Alexander Morgan Capron, LL.B.
Eric J. Cassell, M.D., M.A.C.P.
R. Alta Charo, J.D.
James F. Childress, Ph.D.
David R. Cox, M.D., Ph.D.
Rhetaugh G. Dumas, Ph.D., R.N.
Laurie M. Flynn*
Carol W. Greider, Ph.D.
Steven H. Holtzman
Bernard Lo, M.D.
Lawrence H. Miike, M.D., J.D.
Thomas H. Murray, Ph.D.
William C. Oldaker, LL.B.
Diane Scott-Jones, Ph.D.
*Resigned on May 10, 2001

Ethical and Policy Issues in Research Involving Human Participants; Summary

Protecting Research Participants—A Time for Change

Introduction

Protecting the rights and welfare of those who volunteer to participate in research is a fundamental tenet of ethical research. A great deal of progress has been made in recent decades in changing the culture of research to incorporate more fully this ethical responsibility into protocol design and implementation. In the 1960s and 1970s, a series of scandals concerning social science research and medical research conducted with the sick and the illiterate underlined the need to systematically and rigorously protect individuals in research (Beecher 1966; Faden and Beauchamp 1986; Jones 1981; Katz 1972; Tuskegee Syphilis Study Ad Hoc Advisory Panel 1973). However, the resulting system of protections that evolved out of these rising concerns—although an improvement over past practices—is no longer sufficient. It is a patchwork arrangement associated with the receipt of federal research funding or the regulatory review and approval of new drugs and devices. In addition, it depends on the voluntary cooperation of investigators, research institutions, and professional societies across a wide array of research disciplines. Increasingly, the current system is being viewed as uneven in its ability to simultaneously protect the rights and welfare of research participants and promote ethically responsible research.

Research involving human participants has become a vast academic and commercial activity, but this country's system for the protection of human participants has not kept pace with that growth. On the one hand, the system is too narrow in scope to protect all participants, while on the other hand, it is often so unnecessarily bureaucratic that it stifles responsible research. Although some reforms by particular federal agencies and professional societies are under way,[1] it will take the efforts of both the executive and legislative branches of government to put in place a streamlined, effective, responsive, and comprehensive system that achieves the protection of all human participants and encourages ethically responsible research.

Clearly, scientific investigation has extended and enhanced the quality of life and increased our understanding of ourselves, our relationships with others,